

**COMPARISON OF THE PaCO_2 - EtCO_2 GRADIENT BETWEEN THE
PRE CARDIO PULMONARY BYPASS AND THE POST CARDIO
PULMONARY BYPASS PERIOD IN PATIENTS UNDERGOING
CORONARY ARTERY BYPASS GRAFT.**

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OBSERVATIONAL STUDY

A

**DESSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF
M.D. BRANCH (ANAESTHESIOLOGY) EXAMINATION OF THE
DR. M.G.R.MEDICAL UNIVERSITY TAMILNADU, CHENNAI**

TO BE HELD IN MARCH 2009.

CERTIFICATE

This is to certify that the dissertation entitled “**COMPARISON OF THE PaCO₂-EtCO₂ GRADIENT BETWEEN THE PRE CARDIO PULMONARY BYPASS AND THE POST CARDIO PULMONARY BYPASS PERIOD IN PATIENTS UNDERGOING CORONARY ARTERY BYPASS GRAFT**” is the bonafide original work done by **Dr. KOTHAI.S.** This study was undertaken at the **Christian Medical College Hospital, Vellore** from the year 2007-2009 under my direct guidance and supervision, in partial fulfillment of the requirement for the award of the **M.D. degree (Branch- X) Anaesthesiology of the Tamilnadu Dr.M.G.R. Medical University.**

Guide:

Head of the department:

**Dr.Manickam Ponniah
Professor**

**Dr.Sarah Ninan
Professor and Head**

**Department of Anaesthesia
Christian Medical College And Hospital
Vellore- 632004.
Tamil nadu.**

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PATIENT INFORMATION SHEET

INFORMED CONSENT FORM TO PARTICIPATE IN A CLINICAL TRIAL

PROFORMA

MASTER DATA SHEET

AIM

AIM

1. To study and compare the relationship of PaCO₂- EtCO₂ gradient between pre cardio pulmonary bypass and post cardio pulmonary bypass period.
2. To find out if any factor affects this gradient.

INTRODUCTION

INTRODUCTION

Knowledge and control of arterial CO_2 is an essential part of anaesthesia especially during cardiac surgery. To determine the adequacy of alveolar ventilation it is important to know PaCO_2 . Capnography constitutes a useful and non invasive means of continuously measuring EtCO_2 which usually correlates with PaCO_2 . In normal individuals the arterial- End tidal CO_2 difference (PaCO_2 - EtCO_2 gradient) may vary from 2-5 mm Hg¹⁻⁵. But several factors like hypotension, decrease in pulmonary blood flow can result in alteration in ventilation-perfusion ratio. This in turn, alters the alveolar dead space and affects arterial- alveolar CO_2 difference. During cardio pulmonary bypass there is considerable alterations in ventilation-perfusion ratio and alveolar dead space which may extend into the post cardio pulmonary bypass period. It is important to know the PaCO_2 - EtCO_2 gradient variation during cardiac surgery and identify the factors causing this variation. Hence we decided to study the Arterial – End tidal CO_2 gradient in the pre cardio pulmonary bypass period and compare it to the post cardio pulmonary bypass period in patients undergoing coronary artery bypass graft.

LITERATURE REVIEW

LITERATURE REVIEW

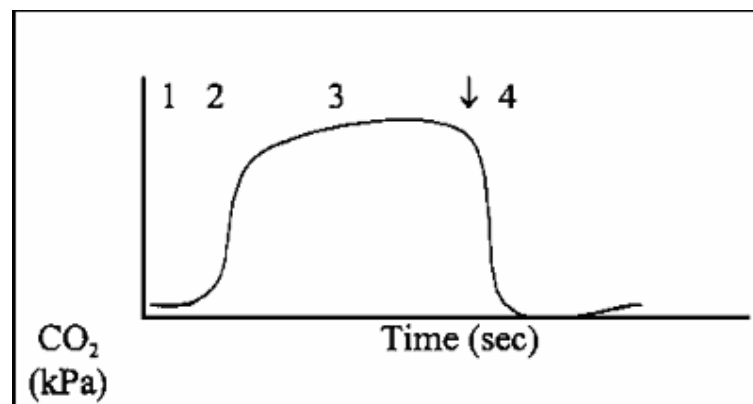
Capnography has become an integral part of monitoring the patient intraoperatively. During anesthesia, capnography directly reflects the elimination of CO₂ by the lungs. Indirectly, it reflects the production of CO₂ by tissues and the circulatory transport of CO₂ to the lungs. Thus capnography is an important non-invasive technique that provides information about CO₂ production, pulmonary perfusion and alveolar ventilation, respiratory patterns as well as elimination of CO₂ by the lungs.

PHYSIOLOGY:

At the end of inspiration, assuming that there is no rebreathing, the airway and the lungs are filled with CO₂-free gases. Carbon dioxide diffuses into the alveoli and equilibrates with the end-alveolar capillary blood ($PACO_2 = PaCO_2 = 40$ mm Hg). The actual concentration of CO₂ in the alveoli is determined by the extent of ventilation and perfusion into the alveoli (V/Q ratio). The alveoli with higher ventilation in relation to perfusion (high V/Q alveoli) have lower CO₂ compared to alveoli with low V/Q ratio that would have higher CO₂. As one moves proximally in the respiratory tract, the concentration of CO₂ falls gradually to zero at some point. The volume of CO₂-free gas is termed respiratory dead space and here there is no exchange of oxygen (O₂) and CO₂ between the inspired gases and the blood. As the patient exhales, a CO₂ sensor at the mouth will detect no CO₂ as the initial gas sampled will be the CO₂-free gas from the dead space. As exhalation continues, CO₂ concentration rises gradually and reaches a peak as the CO₂ rich gases from the alveoli make their way to the CO₂ sensing point at the mouth. At the end of exhalation, the CO₂ concentration

decreases to zero (baseline) as the patient commences inhalation of CO₂ free gases. The evolution of CO₂ from the alveoli to the mouth during exhalation, and inhalation of CO₂ free gases during inspiration gives the characteristic shape to the CO₂ curve which is identical in all humans with healthy lungs.¹ Any deviation from this identical shape should be investigated to determine a physiological or a pathological cause producing the abnormality.

- A typical time capnogram can be considered as two segments and two angles; an inspiratory segment and an expiratory segment, and alpha and beta angles.^{2,3}



Expiratory segment

The expiratory segment of a time capnogram is divided into three phases: I, II, III. Occasionally, at the end of phase III, a terminal upswing, phase IV, may occur.

Phase I

Represents the CO₂-free gas from the airways (anatomical and apparatus dead space).

Phase II

Consists of a rapid S-shaped upswing on the tracing (due to mixing of dead space gas with alveolar gas).

Phase III

Consists of an alveolar plateau representing CO₂-rich gas from the alveoli. It almost always has a positive slope, indicating a rising PCO₂ and is due to the following reasons:

(ii) Steady excretion of CO₂ into the alveoli

Carbon dioxide is being continuously excreted into the alveoli which are becoming progressively smaller as expiration continues. This results in a steady increase in alveolar PCO₂ towards the end of expiration, and hence contributes to a rising positive slope of phase III as expiration proceeds.

(ii) The late emptying of alveoli with lower ventilation/perfusion (V/Q) ratios and, therefore, relatively higher PCO₂.

If all the alveoli had the same PCO₂, then irrespective of the emptying patterns, phase III would be nearly horizontal. However, this ideal situation does not occur, even in normal lungs which have a wide range of V/Q ratios. Some alveoli have a higher V/Q ratio (over ventilated) than ideal alveoli and hence they have a relatively lower PCO₂. Others have a lower V/Q ratio than ideal alveoli (under ventilated) resulting in a relatively higher PCO₂. The delayed emptying of these alveoli with low V/Q (high PCO₂) contributes to the rising slope of phase III. The mechanisms producing this effect are:-^{4, 5}

(A) Within the terminal respiratory unit

Ventilation:perfusion (V/Q) mismatch within the unit may be due either to incomplete gas mixing (alveolar mixing defect) or to the fact that the maximum ventilation and maximum perfusion to that unit are out of sequence in respect to time (temporal mismatching - perfusion is highest during the latter part of expiration when ventilation is lowest). The scatter of V/Q ratios produced as a result is axially distributed with those alveoli having low V/Q ratio (higher PCO₂) being distributed distally and emptying later.

(B) Between respiratory units

There may be a regional variation in ventilation per unit perfusion producing a spectrum of V/Q ratios (spatial mismatching). Under these circumstances, the slope of phase III is determined by the nature of emptying of the alveolar units: synchronous or asynchronous. If the units empty

synchronously, the gas from well-perfused and underperfused alveoli is expired simultaneously, resulting in a horizontal phase III or else a phase III with minimal slope. However, if the units empty asynchronously, units with longer time constants, hence higher PCO_2 , would empty later (sequential emptying) resulting in a rising slope of Phase III. The slope of the phase III is dependent, therefore, on the emptying patterns of various alveoli with different V/Q ratios as well as continuous CO_2 excretion into the alveoli. The relative contributions of all of the above mechanisms cannot be separated and all occur simultaneously, influencing the height or the slope of phase III. Factors, such as changes in cardiac output, CO_2 production, airway resistance and functional residual capacity (FRC) may further affect the V/Q status of the various units in the lung, and thus influence the height or the slope of phase III. This attribute makes capnography a useful diagnostic tool to detect abnormalities in ventilation perfusion mismatch of the lung.

Inspiratory segment - Phase 0

After phase III is complete, the descending limb makes an almost right angle turn and rapidly descends to the baseline. This represents the inspiratory phase during which the fresh gases (CO_2 -free gases) are inhaled and CO_2 concentration falls rapidly to zero.^{2,3} The segment of the CO_2 trace from the beginning of inspiration to the beginning of expiration, which includes the descending limb and the initial part of the horizontal baseline, can be designated as phase 0. The later part of the horizontal baseline is the phase I of expiratory segment. Phase 0 represents dynamics of inspiration.

Alfa Angle

The angle between phases II and III, which has been referred to as the alpha angle, increases as the slope of phase III increases. The alpha angle (primarily linked to variations in time constants within the lung) is thus an indirect indication of V/Q status of the lung.⁶

Beta Angle

The nearly 90 degrees angle between phase III and the descending limb in a time capnogram has been termed as the beta angle.⁷ This can be used to assess the extent of rebreathing.⁷ During rebreathing, there is an increase in beta angle from the normal 90 degrees. As rebreathing increases, the horizontal baseline of phase 0 and phase I can be elevated above normal.⁷ Occasionally, other factors, such as prolonged response time of the capnometer compared to respiratory cycle time of the patient, particularly in children, can produce increase in the beta angle with the elevation of the baseline of phase 0 and phase I, as observed in rebreathing.⁸

PEtCO₂ as a measure of PaCO₂

Measurement of End tidal carbon dioxide constitutes the useful non-invasive measurement of monitoring PaCO₂ and hence the ventilatory status of the patient during anaesthesia.

The advantages of capnography as a measure of PaCO₂ are:

- (1) It provides continuous estimate of PaCO₂.
- (2) It gives an instantaneous estimate of PaCO₂.

(3) After the initial capital expense of purchasing the equipment it is inexpensive to use.

In a study comparing PaCO_2 and PEtCO_2 during anaesthesia, the difference between these values correlated with the presence of lung disease, age, ASA class and systolic blood pressure.

But other studies have shown that changes in PaCO_2 were inaccurately predicted by PEtCO_2 .

In summary, the disadvantage of using EtCO_2 to estimate PaCO_2 is that there is an arterial to end tidal CO_2 difference which is dependent on various factors.⁹⁻¹²

P (a-ET) CO_2 gradient

Under normal circumstances, the PETCO_2 (the CO_2 recorded at the end of the breath which represents PCO_2 from alveoli which empty last) is lower than PaCO_2 (average of all alveoli) by 2-5 mmHg, in adults.^{1-5, 13, 14} The (a-ET) (arterial- End tidal) PCO_2 gradient is due to the V/Q mismatch in the lungs (alveolar dead space) as a result of temporal, spatial, and alveolar mixing defects. In healthy children, the (a-ET) PCO_2 gradient is smaller (-0.65-3 mm Hg) than in adults.¹⁴⁻¹⁹ This is due to a better V/Q matching, and hence a lower alveolar dead space in children than in the adults.⁹ The (a-ET) PCO_2 / PaCO_2 fraction is a measure of alveolar dead space, and changes in alveolar dead space correlate well with changes in (a-ET) PCO_2 .⁵ An increase in (a-ET) PCO_2 suggests an increase in dead space ventilation. Hence (a-ET) PCO_2 is an indirect estimate of V/Q mismatching of the lung.

However, (a-ET) PCO_2 does not correlate with alveolar dead space in all circumstances. Changes in alveolar dead space correlate with (a-ET) PCO_2 only when phase III is flat or has a minimal slope. However, if phase III has a steeper slope, the terminal part of phase III may intercept the line representing PaCO_2 , resulting in either zero or negative (a-ET) PCO_2 even in the presence of alveolar dead space. Therefore, the (a-ET) PCO_2 is dependent both on alveolar dead space as well as factors that influence the slope of phase III. This implies that an increase in the alveolar dead space need not be always being associated with an increase in the (a-ET) PCO_2 . The (a-ET) PCO_2 may remain the same if there is an associated increase in the slope of the phase III. For example, it has been observed during cardiac surgery that alveolar dead space was increased at the end of cardiopulmonary bypass but as the slope of phase III was also increased, there was no change in (a-ET) PCO_2 .^{21, 22}

Factors affecting (a-Et) PCO_2

This difference can vary from patient to patient and is dependent on various factors. (a-ET) PCO_2 increases with age, emphysema, and in circumstances where alveolar dead space increases such as in low cardiac output states, hypovolemia, and pulmonary embolism and anaesthesia itself. The difference increases with large tidal volumes and low frequency ventilation.

Cardiac output and (a-ET) PCO_2

Reduction in cardiac output and pulmonary blood flow result in a decrease in PETCO_2 and an increase in (a-ET) PCO_2 .^{23,24} The percent decrease in PETCO_2 directly correlated with the percent decrease in cardiac output (slope=

0.33, $r^2=0.82$ in 24 patients undergoing aortic aneurysm surgery with constant ventilation).²⁵ Also, the percent decrease in CO_2 elimination correlated with the percent decrease in cardiac output similarly (slope=0.33, $r^2=0.84$).²⁵ The changes in PETCO_2 and CO_2 elimination following hemodynamic perturbation were parallel. These findings suggest that decrease in PETCO_2 quantitatively reflect the decreases in CO_2 elimination.²⁵

Increases in cardiac output and pulmonary blood flow result in better perfusion of the alveoli and a rise in PETCO_2 .^{23, 24} Consequently alveolar dead space is reduced as is (a-ET) CO_2 . The decrease in (a-ET) PCO_2 is due to an increase in the alveolar CO_2 with a relatively unchanged arterial CO_2 concentration, suggesting better excretion of CO_2 into the lungs. The improved CO_2 excretion is due to better perfusion of upper parts of the lung.²⁴ Relationship between PETCO_2 and pulmonary artery blood flow was studied during separation from cardiopulmonary bypass²⁶ This showed that PETCO_2 is a useful index of pulmonary blood flow. A PETCO_2 greater than 30 mm Hg was invariably associated with a cardiac output more than 4 L/min or a cardiac index > 2 L/min.²⁶ Furthermore, when PETCO_2 exceeded 34 mm Hg, pulmonary blood flow was more than 5 L/min ($\text{CI} > 2.5$ L).²⁶

Thus, under conditions of constant lung ventilation, PETCO_2 monitoring can be used as a monitor of pulmonary blood flow.²⁶⁻³¹

Recently, using Fick's Principle, attempts were made to determine cardiac output non-invasively implementing periods of CO_2 rebreathing during which CO_2 partial pressure of oxygenated mixed venous blood was obtained from the measured exponential rise of the PETCO_2 value. In addition, oxygen uptake,

carbon dioxide elimination, end-tidal PCO_2 , oxygen saturation, and tidal volume were determined. The results are encouraging in patients with healthy lungs.³² Whereas the results are controversial when the lungs are diseased.³³

BLOOD PRESSURE AND P (a-ET) CO_2

A study of seven anaesthetized patients whose mean arterial pressure (MAP) was lowered from 67 mmHg to an average of 50 mmHg, showed that physiological dead space increased in all patients. The increase resulted from enlargement of both anatomical and physiological dead space especially the later.³⁴ In a later study done by the same investigator it was shown that the increase in P (a-ET) CO_2 correlated strongly with the decrease in pulmonary artery pressure (PAP). Since a fall in pulmonary artery pressure parallels a reduction in MAP, the same effect on P (a-ET) CO_2 is seen with reductions in MAP. Hypovolemia by its effect on cardiac output, MAP and PAP can lead to increase in P (a-ET) CO_2 .

P (a-ET) CO_2 AND VENTILATION

Normal spontaneous ventilation results in preferential distribution of ventilation to dependent areas in the upright, lateral, supine and prone positions.³⁵ Froese and Bryan found that unlike in patients breathing spontaneously, paralysis and mechanical ventilation leads to preferential displacement of the passive diaphragm in the non- dependent zones where airway pressure and perfusion are least thus leading to an increase in dead space.³⁶ It has been shown that anaesthesia leads to increase in the physiological dead space.³⁷

Russell et al., studying the stability of arterial to end tidal CO₂ gradients during post operative cardio- respiratory support found that though changing respiratory rate and FiO₂ can change V/Q ratios a correlation could not be demonstrated with P (a-ET) CO₂.³⁸

In a very early study Nunn et al., concluded that P (a-ET) CO₂ was not related to tidal volume.³⁹

Ramer et al., measuring the EtCO₂ during anaesthesia found no consistent correlation between P (a-ET) CO₂ gradient and variation in ventilation.³⁹

P (a-ET) CO₂ AND ANAESTHESIA

During anaesthesia, increased degrees of uneven ventilation / perfusion relationships can occur. It has been found that a considerable impairment of the lung function exists during anaesthesia. This is expressed by increase in Arterial to Alveolar CO₂ difference.⁴⁰

This increased difference is attributed to various factors:

- (1) Partial collapse of the lungs during continuous controlled ventilation without intermittent deep breath.⁴¹
- (2) Arterial hypotension.⁴¹
- (3) High FiO₂ leading to absorption atelectasis thus increasing shunt.⁴¹
- (4) Reduction in Functional residual capacity.⁴²

Severinghaus et al., showed that major part of this difference is mainly due to grossly reduced perfusion in parts of lung that is by an increased in alveolar dead space.⁴³

Ramwell et al., found the difference between Arterial to End tidal CO₂ to be significantly greater during anaesthesia⁴⁴

PaCO₂-PETCO₂ GRADIENT IN CABG

R.Fletcher et al., studied 43 patients undergoing CABG surgery and found that the PaCO₂-PETCO₂ gradient varied widely.^{45, 46} The possible mechanisms they had attributed were

- (1) Decreased alveolar perfusion secondary to reduced cardiac output.
- (2) Altered patterns of pulmonary filling and emptying secondary to changes in functional residual capacity and the shape of the thoracic cavity brought about by sternotomy, sternal closure and extra corporeal circulation.
- (3) Increased intra pulmonary shunting by atelectasis, fluid overload and airway disease.

Myles PS et al., studied PaCO₂-PEtCO₂ gradient continuously during cardiac surgery and found that there was a significant change in the gradient after cardio pulmonary bypass.⁴⁷

Opper SE et al., studied the effect of oxygenator and bypass flow pattern on PaCO₂-PEtCO₂ gradient and found that there was a trend for the gradient to increase in the post bypass period though it was not statistically significant.

⁴⁸They also found that changes in mean arterial pressure, cardiac index, systemic vascular resistance and the duration of bypass did not influence the PaCO₂-PEtCO₂ gradient.

MATERIALS & METHODOLOGY

MATERIALS AND METHODOLOGY

We did a preliminary study on ten patients who underwent elective CABG surgery. The $\text{PaCO}_2\text{-EtCO}_2$ gradient was calculated in pre bypass and post bypass period.

The main purpose of this preliminary study was

- (1) To study and compare the relationship of Arterial CO_2 - End tidal CO_2 gradient between the pre cardio pulmonary bypass and post cardio pulmonary bypass period.
- (2) To calculate the sample size required for the study.

SAMPLE SIZE

From the preliminary study, the sample size was calculated in order to derive statistically significant conclusions.

The required sample size to show a significant change in $\text{PaCO}_2\text{-EtCO}_2$ gradient in pre cardio pulmonary and post cardio pulmonary bypass period was found to be 33 subjects with 80% power and at 5% level of significance.

THE MAIN STUDY

INCLUSION CRITERIA

All the patients undergoing elective CABG surgery.

Patients willing to participate in the study.

EXCLUSION CRITERIA

Patients with severe pulmonary disease ($FEV_1 \leq 50\%$ of predicted value).

Patients not willing to participate in the study.

METHOD OF STUDY

All the patients undergoing elective CABG were visited pre operatively, the nature of the study explained to them and written consent taken.

They were pre medicated with Tab. Diazepam 5 mg the night before surgery and Tab. Diazepam 7.5 mg 30 min before induction. They were shifted to the operating room with O₂ mask 6 Liters/min in propped up position. Monitoring was established with pulse oximeter and ECG. A peripheral venous catheter was inserted, 500 ml of normal saline infusion started. Radial artery cannulation was done subsequently for invasive arterial pressure monitoring.

After pre oxygenation with 100% O₂, Anaesthesia was induced with Inj. Midazolam 0.05-0.1 mg/kg; Inj. Fentanyl 2-3 mcg/kg and inhalational anaesthetic Sevoflurane 2-6 % conc (or) Isoflurane 2-3% conc. Patients were paralyzed with Vecuronium 0.1 mg/kg (or) Pancuronium 0.1 mg/kg and ventilated

for maximum of 5 minutes. Hypotension during this period was managed with small bolus doses of Inj. Phenylephrine 25-50 mcg.

Endotracheal intubation was done and bilateral air entry checked. A central venous catheter was inserted through Right Internal jugular vein for central venous pressure monitoring and for administration of inotropes. A nasopharyngeal temperature probe was inserted to measure the temperature. As a protocol of this institution, adrenaline infusion was started at 0.1 mcg/kg up to 0.2 mcg/kg in the post bypass period.

Ventilation was maintained with

Datex Ohmeda 134 ventilator

Mode : Volume controlled mode with values set at

Respiratory rate : 12/min

Tidal volume : 8 ml/kg

Extra corporeal circulation was maintained with cardio pulmonary bypass machine. Fluid balance was maintained with central venous pressure monitoring.

ESTIMATION OF PaCO₂ AND ETCO₂

End tidal carbon dioxide was continuously monitored in all patients by a side stream infra red analyzer .The arterial blood PaCO₂ measurement was calculated in a heparinised 2 ml syringe from the arterial line after discarding 5 ml of aspirate and made air tight before sending for analysis.

SAMPLING REGIMEN

Measurements were made at four stages during surgery.

1. 15 min after intubation (to allow time for sufficient equilibration).
2. 5 min after sternotomy with the sternum fully retracted, before cannulation of the heart.
3. 5 min after Cardio pulmonary bypass, with the heart decannulated and the sternum is fully retracted.
4. 5 min after sternal closure.

PARAMETERS MEASURED

Hemodynamic parameters:

Heart rate (beats/min)
Mean arterial pressure (mmHg)
Central venous pressure (mmHg)

Ventilatory parameters:

End tidal CO₂ (mm Hg)
Peak inspiratory pressure (CmH₂O)
Expiratory tidal volume/ Inspiratory tidal volume (ml)
PEEP (CmH₂O)

Arterial blood gas analysis:

FiO₂ (%)
PaO₂ (mmHg)
PaCO₂ (mmHg)
PaCO₂/FiO₂ Ratio
PaCO₂- EtCO₂ gradient
Temperature (° c)

All the information were recorded in the proforma and analyzed with the help of CMC statistician.

RESULTS

RESULTS

Figure 1: Distribution of Age

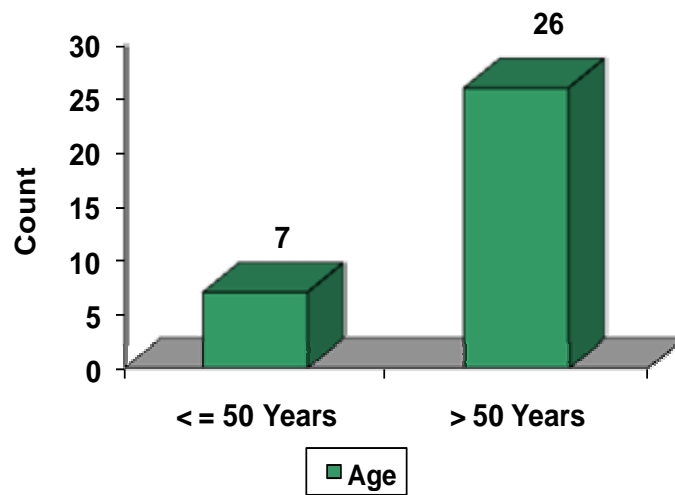
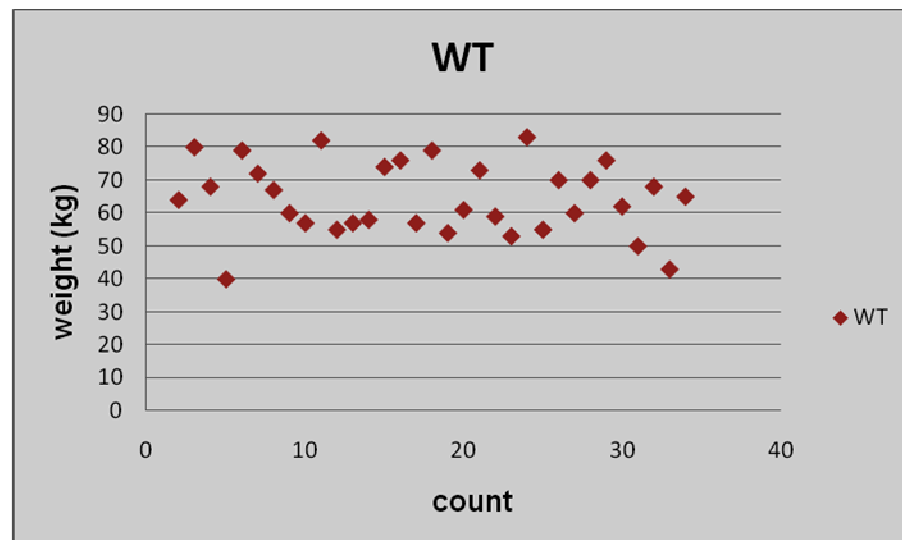


Figure 2: Distribution of weight



Thirty three patients who underwent elective Coronary Artery Bypass Grafting surgery were included in the study.

Age and weight Distribution

The age and weight distribution are displayed in figure1 and 2 respectively. The age distribution is between 30 and 72years.out of 33 patients seven patients were ≤ 50 years and 26 were above 50 years.The weight distribution is between 43 and 83kgs.

Figure 3: SEX DISTRIBUTION

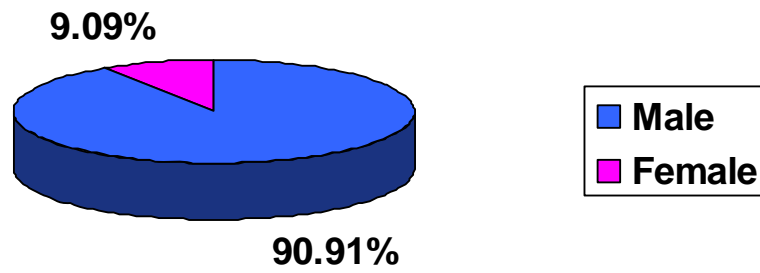
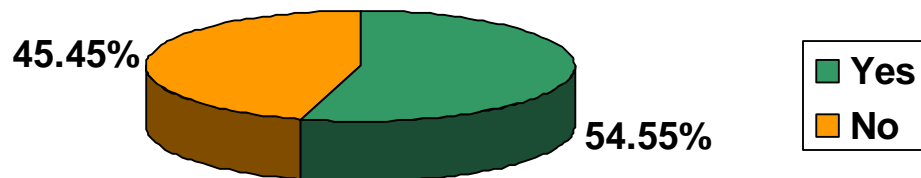


Figure 4: SMOKING STATUS DISTRIBUTION



Sex distribution which is shown in figure 3 shows that out of 33 patients 90.91% (30) were males and the remaining 9.09% (3) were females.

The smoking status distribution is displayed in figure 4 which is showing out of 33 patients 54.55 %(18) were smokers.

Table 1: PaCO₂-EtCO₂ gradients in pre cardio pulmonary bypass and post cardio pulmonary bypass period

PaCO₂-EtCO₂ gdt	Pre- CPB	Post-CPB
Mean	4.5667	8.1985
Standard deviation	1.36997	3.36144
Standard error mean	238.48	.58515

Table 2: PaCO₂-EtCO₂ gradient difference

PaCO₂- EtCO₂ gdt difference	Mean	Standard deviation	Standard error mean	“t”	Significance
	-3.63182	3.12149	.54338	-6.684	.000

The PaCO₂- EtCO₂ gradients in pre cardiopulmonary bypass and post cardio pulmonary bypass period and the difference are shown in table 1 & 2 respectively. The mean pre cardio pulmonary bypass (CP Bypass) PaCO₂ – EtCO₂ gradient was 4.5667 and the mean post CP Bypass PaCO₂ – EtCO₂ gradient was 8.1985. There is statistically significant (p=.000) increase in the gradient (mean of 3.6318) after cardio pulmonary bypass using paired “t” test.

Table 3: PaCO₂ in pre and post cardio pulmonary bypass period

PaCO₂	Pre-CPB	Post-CPB
Mean	35.7152	42.2758
Standard deviation	4.08663	5.06446
Standard error mean	.71139	.88161

Table 4: PaCO₂ difference

PaCO₂ difference	Mean	Standard deviation	Standard error mean	“t”	Significance
	-6.56061	3.80143	.66174	-9.914	.000

Table 3 & 4 shows PaCO₂ in pre cardiopulmonary bypass and post cardio pulmonary bypass period and the difference respectively. The mean pre CP Bypass PaCO₂ was 35.7152 and the mean post CP Bypass PaCO₂ was 42.2758. There is a statistically significant (p=.000) increase in PaCO₂ after cardio pulmonary bypass (mean of 6.5606).

Table5: EtCO₂ in pre and post cardio pulmonary bypass period

EtCO₂	Pre-CPB	Post-CPB
Mean	31.2424	34.1364
Standard deviation	3.92134	3.85331
Standard error mean	.68262	.67078

Table 6: EtCO₂ difference

EtCO₂ difference	Mean	Standard deviation	Standard error mean	“t”	Significance
	-2.89394	3.96027	.68939	-4.198	.000

Table 5 & 6 shows EtCO₂ in pre cardiopulmonary bypass and post cardiopulmonary bypass period and the difference respectively. . The mean pre CP Bypass EtCO₂ was 31.2424 and the mean post CP Bypass EtCO₂ was 34.1364. There is a statistically significant (p=.000) increase in EtCO₂ after cardiopulmonary bypass (mean of 2.8939).

Table 7: Heart rate in pre and post cardio pulmonary bypass period

Heart rate	Pre CPB	Post CPB
Mean	74.3636	95.7727
Standard deviation	15.43397	12.97900
Standard error mean	2.68671	2.25935

Table 8: Heart rate difference

Heart rate difference	Mean	Standard deviation	Standard error mean	“t”	Significance
	- 2.14091E1	17.00710	2.96056	-7.231	.000

Mean Heart rate in pre and post cardio pulmonary bypass periods are shown in tables 7 &8 respectively. . The mean pre CP Bypass heart rate was 74.3636 and the mean post CP Bypass heart rate was 95.7727. There is a statistically significant ($p=.000$) increase in the heart rate after cardio pulmonary bypass (mean of 2.1409).

Table 9: Mean Arterial Pressure in Pre and Post cardio pulmonary bypass period.

Mean Arterial Pressure	Pre-CPB	Post-CPB
Mean	72.1364	65.6667
Standard deviation	8.63882	6.92332
Standard error mean	1.50383	1.20519

Table 10: Mean Arterial Pressure difference.

Mean Arterial Pressure difference	Mean	Standard deviation	Standard error mean	“t”	Significance
	6.46970	9.65655	1.68099	3.849	.001

There is a statistically significant ($p=.001$) decrease in mean arterial pressure (mean of 6.469) following cardio pulmonary bypass. (Tables 9 & 10). The mean pre CP Bypass mean arterial pressure was 72.1364 and the mean post CP Bypass mean arterial pressure was 65.6667.

Table 11: Central Venous Pressure in Pre and Post cardio pulmonary bypass period.

Central Venous Pressure	Pre-CPB	Post-CPB
Mean	4.6061	5.0000
Standard deviation	1.93147	2.59507
Standard error mean	.33623	.45174

Table 12 : Central Venus Pressure Différence.

Central Venous Pressure difference	Mean	Standard deviation	Standard error mean	“t”	Significance
	-.39394	2.46145	.42848	-.919	.365

There is no statistically significant change in Central Venous Pressure in the post cardio pulmonary bypass period compared to the pre bypass values. (Tables 11& 12).The mean pre CP Bypass central venous pressure was 4.6061 and the mean post CP Bypass central venous pressure was 5.0000.

Table 13: Peak Inspiratory Pressure in Pre and Post cardio pulmonary bypass period.

Peak Inspiratory pressure	Pre-CPB	Post-CPB
Mean	13.4545	13.5606
Standard deviation	2.29593	2.48671
Standard error mean	.39967	.43288

Table 14: Peak Inspiratory pressure difference

Peak Inspiratory pressure difference	Mean	Standard deviation	Standard error mean	“t”	Significance
	-.10606	1.73546	.30211	-.351	.728

There is no statistically significant change in Peak inspiratory Pressure in the post cardio pulmonary bypass period compared to the pre bypass values. (Tables 13& 14). The mean pre CP Bypass mean peak inspiratory pressure was 13.4545 and the mean post CP Bypass peak inspiratory pressure was 13.5606.

Table 15: PaO₂/ FiO₂ Ratio in Pre and Post cardio pulmonary bypass period.

PaO₂/FiO₂ Ratio	Pre-CPB	Post-CPB
Mean	404.7455	376.1939
Standard deviation	67.15693	91.49615
Standard error mean	11.69052	15.92744

Table 16: PaO₂/FiO₂ Ratio difference

PaO₂/FiO₂ Ratio difference	Mean	Standard deviation	Standard error mean	“t”	Significance
	28.55152	83.83542	14.59387	1.956	.059

There is no statistically significant change in $\text{PaO}_2/\text{FiO}_2$ ratio in the post cardio pulmonary bypass period compared to the pre bypass values. (Tables 15& 16). The mean pre CP Bypass $\text{PaO}_2/\text{FiO}_2$ ratio was 404.7455 and the mean post CP Bypass $\text{PaO}_2/\text{FiO}_2$ ratio was 376.1939.

Table 17: Temperature in Pre and Post cardio pulmonary bypass period.

Temperature	Pre-CPB	Post-CPB
Mean	35.8515	36.2758
Standard deviation	.55783	.54703
Standard error mean	.09710	.09523

Table 18: Temperature difference

Temperature difference	Mean	Standard deviation	Standard error mean	“t”	Significance
	-.42424	.60443	.10522	-4.032	.000

Mean temperature in pre and post cardio pulmonary bypass are shown in tables 17 & 18.) The mean pre cp bypass temperature was 35.8515 and the mean post CP Bypass temperature was 36.2758. There is a statistically significant ($p=.000$) increase in temperature after CP Bypass (mean of .424).

Table 19: Correlation between PaCO₂-EtCO₂ gradient differences with other variables.

	Pearson correlation	Significance
Heart rate difference	-.019	.916
Mean Arterial Pressure difference	.148	.411
Central Venous Pressure difference	-.046	.799
Peak Inspiratory Pressure difference	.242	.174
PaO ₂ / FiO ₂ Ratio difference	.073	.688
Temperature difference	-.333	.058

Table 19 shows the correlation between $\text{PaCO}_2\text{-EtCO}_2$ gradient difference (Pre cardio pulmonary bypass _ Post cardio pulmonary bypass period) and Mean Heart rate difference, Mean arterial pressure difference, Central venous pressure difference, Peak inspiratory pressure difference, $\text{PaO}_2/\text{FiO}_2$ Ratio difference and Temperature difference. There is no statistically significant correlation between any of the variables.

$\text{PaCO}_2\text{-EtCO}_2$ gradient in smokers was 8.47 and in non smokers it was 8.07.

DISCUSSION

DISCUSSION

It is known that the difference between PaCO_2 and EtCO_2 is about 2-5 mmHg even in normal individuals.¹⁻⁵ It was postulated that the gradient between PaCO_2 and EtCO_2 varies widely during open-heart surgery especially before and after the cardio pulmonary bypass (CP Bypass) period due to various factors.^{45,46} We were interested in studying if a change in the PaCO_2 - EtCO_2 gradient takes place in the post-bypass period during CABG surgery, and if it takes place, to quantify the magnitude of the change in the gradient and try to identify any factor affecting this gradient.

On studying 33 patients, one of the major positive observations was the increase in PaCO_2 - PEtCO_2 gradient in the post cardio-pulmonary bypass period compared to pre bypass period. This increase in gradient is from as small as 0.6 to a maximum of 15.7 with mean of 3.6.

Even though both the absolute values of PaCO_2 and EtCO_2 increased in the post bypass period significantly, the magnitude of PaCO_2 rise is more than that of EtCO_2 ; thus resulting in a greater gradient.

While trying to identify the causative or contributory factors that caused a greater increase in PaCO_2 over EtCO_2 , this study was able to identify one definite factor.

This is the body temperature of the patient which increased significantly in the post-cardiopulmonary bypass period compared to the pre bypass period. This statistically significant increase in the temperature had a positive correlation with

the increase in the gradient. From the time the patient arrives in the efficiently air conditioned operating room, till the time the patient is hooked onto the bypass machine, there is a gradual drop in body temperature; mainly because, deliberately, no active warming of the patient is performed. Before coming-off CP Bypass, active rewarming of the patient's blood is instituted to such an extent that the temperature very often overshoots above normal. Large increase in carbon dioxide production due to increase in temperature during rewarming most likely affects this increase in the PaCO_2 - EtCO_2 gradient in the post cardio pulmonary bypass period.

The other major factors which could be responsible for this increase in the gradient are the following, which are specific to the post CP Bypass period and are brought on by a combination of events that occur during CP Bypass.

- (1) Increase in the dead space due to decrease in cardiac output.
- (2) Increase in intra pulmonary shunting because of micro (or) macroatelectasis.
- (3) Interstitial fluid accumulation in the lungs

All the above factors could interfere with or have an impact on the gas exchange in the alveoli.⁴⁹

1) Increase In Dead Space Due To Decrease In Cardiac Output

Cardiac output was not directly measured in this study; but the statistically significant decrease in the mean arterial blood (MAP) pressure recorded in this study does probably reflect decrease in cardiac output. The

myocardium does suffer some insult during the CP Bypass period to result in the decrease in MAP in the post CPB period. So, though there was no statistically significant correlation between decrease in MAP and the increase in the PaCO₂ and PEtCO₂ gradient, the drop in MAP (statistically significant) probably contributed to the increase in dead space and its effect on the CO₂ gradient. Additionally all patients were started on inotropic support (Inj.Adrenaline 0.1 mcg/kg/min) during weaning from bypass as is our protocol in our institution. This could have caused some element of pulmonary vasoconstriction which also could have a role in the increase in dead space.⁵⁴

The increase in CO₂ production during rewarming which induces a rise in PaCO₂ does not result in the same increase in ETCO₂ because probably the above-mentioned increase in dead space affects CO₂ exchange.

2) Increase In Intra Pulmonary Shunting Because Of Micro (Or) Macroatelectasis

It was believed that atelectasis, whether micro or macro could be a very important factor responsible for the increase in CO₂ gradient; but this study could not demonstrate any evidence in support of this surmise. In fact, there was no statistically significant difference in the PaO₂-FiO₂ ratio between the pre and post bypass period. Also, there was no significant change in the peak airway pressure detected in the post CP Bypass period. Any presence of significant atelectasis would have resulted in decrease in PaO₂/FiO₂ ratio and increase in the peak airway pressure.

If there are varying degrees of atelectasis there can be a slow upstroke of Phase II of the capnogram. If there was some way of measuring the speed of the upstroke in Phase II, the presence or absence of atelectasis could have been confirmed.⁵²

(3) Interstitial Fluid Accumulation In The Lungs

Increased interstitial fluid in the lungs may be an expected accompaniment of CP Bypass. The presence of this feature can explain the increased CO₂ gradient. Once again we failed to show any evidence like increased peak airway pressure or an increased PaO₂/FIO₂ ratio following CP Bypass in this study to suggest that increased interstitial edema was the causative factor.

R.Fletcher et al did a similar study in 43 patients undergoing CABG surgery. They found that the PaCO₂-PETCO₂ gradient varied widely.^{45, 46} Although there were no significant change pre and post CP Bypass period, there were, however, some moderate individual changes. The possible mechanisms they have attributed were

- (1) Decreased alveolar perfusion secondary to reduced cardiac output.
- (2) Altered patterns of pulmonary filling and emptying secondary to changes in functional residual capacity and the shape of the thoracic cavity brought about by sternotomy, sterna closure and extra corporeal circulation.
- (3) Increased intra pulmonary shunting by atelectasis, fluid overload and airway disease.

We applied the Pearson test of correlation to the increase in PaCO_2 - EtCO_2 gradient with the a few other parameters like heart rate, central venous pressure, peak inspiratory pressure etc measured in pre cardio pulmonary bypass and post cardio pulmonary bypass periods.

We found that though there was a statistically significant increase in heart rate in the post cardio pulmonary bypass period compared to the pre bypass value, there was no significant correlation with the increase in PaCO_2 - EtCO_2 gradient in post cardio pulmonary bypass period. This increase in heart rate is probably due to the inotrope (adrenaline) administered in the immediate post bypass period.

When we looked at Central Venous pressure and Peak inspiratory pressure in the pre and post cardio pulmonary bypass period we could not find any significant change which correlated with the increase in the PaCO_2 - EtCO_2 gradient.

In our study, surprisingly and contrary to our expectations we could not find any correlation between the smoking status of the subject and the increase in PaCO_2 - EtCO_2 gradient. This may be because we had excluded patients with significant COPD from this study.

Since the $\text{PaCO}_2 - \text{EtCO}_2$ gradient varies in the post cardio pulmonary bypass period; it is not advisable to depend only on the continuous EtCO_2 value to determine the adequacy of ventilation. The ventilatory parameters that may have been employed in the pre bypass period may not result in the same arterial carbon dioxide in the post bypass period if only the EtCO_2 is used to determine

the ventilatory adequacy. It is advisable to corroborate with the arterial blood gas estimation to determine whether ventilator settings may have to be altered to ensure adequacy of ventilation. This is particularly important when you wish to avoid respiratory acidosis induced worsening of pulmonary hypertension in the post CP Bypass period.

It would be interesting to apply the study in children with congenital heart disease coming for correction under CP Bypass and also in adults with pulmonary hypertension coming for valvular heart surgery; to see what happens to the $\text{PaCO}_2 - \text{EtCO}_2$ gradient.

CONCLUSION

CONCLUSION

This study has demonstrated that there is an increase in the $\text{PaCO}_2/\text{EtCO}_2$ gradient in the post CP Bypass period as compared to the pre CP Bypass time. The increase in the body temperature during weaning from CP Bypass correlates with the increase in gradient. The increased CO_2 production results in an increase in PaCO_2 ; but this does not result in a parallel increase in EtCO_2 probably because of the increase in dead space induced by decrease in cardiac output in the post CP Bypass period. The occurrence of increased CO_2 gradient in the post CP Bypass period suggests that we should corroborate ETCO_2 monitoring with arterial blood gas estimation to see the adequacy of ventilation when fixing ventilator settings to avoid possible respiratory acidosis.

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APPENDIX

APPENDIX

PATIENT INFORMATION SHEET

We, from the Department of Anesthesia in coordination with the Dept.of Cardiothoracic surgery in CMC Vellore, are conducting an observational study in patients undergoing elective CABG(Coronary Artery Bypass Grafting) with assistance of a heart lung machine.

As you are enrolled in the above mentioned study, I would like you to have some information regarding the study conducted.

As you might already be knowing from your doctor, CABG is the surgery done for patients suffering from heart attack which is nothing but blockage of the blood vessels supplying the heart causing heart muscle death.

In this surgery, the blocked vessels are bypassed with healthy vessels from elsewhere. During this surgery heart is completely stopped and blood circulation is maintained using a heart lung machine. This surgery is done under general anesthesia and you will be ventilated with a small tube through your windpipe. During anesthesia to know the adequacy of gas exchange we need to monitor carbon dioxide content in your blood and exhaled gas.

Normally the carbon dioxide content in the arterial blood will be 5mm of mercury less than the exhaled gas. But during CABG the difference between arterial and exhaled carbon dioxide varies widely because of using heart lung machine, artificial ventilation and various other factors.

In this study, we are looking at the variations of the difference between arterial and exhaled carbon dioxide in the pre and post cardiopulmonary bypass period, to find out the factors responsible for the variation and to take necessary steps to reduce this variation.

If you volunteer for this study, during surgery we will take 2 ml of blood from your artery at 4 timed intervals and your exhaled gases will be analyzed for carbon dioxide. However there will be neither extra cost nor momentary benefit to you. Also there will be no discomfort to you as you will be under anaesthesia. There will be a senior anaesthetist supervising this study and you will be monitored continuously.

You will have to understand that your participation in the study is voluntary and that you are free to withdraw at any time, without giving any reason, without your medical care or legal rights being affected.

Informed Consent form to participate in a clinical trial

Study Title: To study the relationship of arterial CO₂ to End tidal CO₂ in the pre cardio- pulmonary bypass and post Cardio- pulmonary bypass period

Study Number: _____

Subject's Name: _____

Date of Birth / Age: _____

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [y]

(ii) I understand that my participation in the study is voluntary and that I am

free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [y]

(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published. [y]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s) [y]

(v) I agree to take part in the above study. [y]

Signature (or Thumb impression) of the Subject: _____

Date: ____/____/____

Signatory's Name: _____

Signature of the Investigator: _____

Date: ____/____/____

Study Investigator's Name: _____

Signature of the Witness: _____

Date: ____/____/____

Name of the Witness: _____

PROFORMA

NAME :
AGE : yrs
SEX : M / F
HOSPITAL NUMBER :
HT : cms
WT : kg
ASA GRADE :
SMOKING STATUS : Y / N
PROPOSED SURGERY :
DURATION OF CPB : Hrs
INOTROPE : Y / N

PARAMETERS	PREBYPASS PERIOD		POST BYPASS PERIOD	
	15 min post intubation	5 min post sternotomy	5 min post CBP	5 min poststernal closure
HR(beats/min)				
MAP(mm/hg)				
CVP(mm/hg)				
Peak ins pr/ Mean pr (CmH ₂ O)				
EtCO ₂ (mm/hg)				
FiO ₂ (%)				
PaO ₂ (mm/hg)				
P/F RATIO				
PaCO ₂ (mm/hg)				
PaCO ₂ -EtCO ₂ gdt				
Temp(⁰ c)				
Min ventilation(L/Min)				
Tidal volume - Insp/Exp(ml)				
PEEP (Cm H ₂ O)				

MASTER DATA SHEET

S.NO	HOS.NO	AGE	SEX	WT	SM	HR(BEATS/MIN)				MAP(MM/HG)				CVP(MM/HG)				PEAK/MEAN INSP PR(CmH2O)			
						PRE		POST		PRE		POST		PRE		POST		PRE		POST	
1	166365D	65	M	64	N	71	77	72	71	60	90	66	64	9	4	11	11	12	16	13	17
2	846976C	50	M	80	Y	94	79	91	96	62	88	69	75	7	6	7	10	17	22	19	22
3	185679D	50	M	68	Y	69	79	129	121	65	78	62	66	4	1	2	4	13	12	13	16
4	173064D	59	M	40	Y	74	75	93	98	72	75	50	52	1	3	1	3	8	8	8	10
5	219250D	56	M	79	N	115	92	85	96	54	72	55	58	2	2	8	9	8	13	14	17
6	193760D	41	M	72	N	56	50	86	93	68	82	68	67	5	5	1	2	17	16	12	17
7	696761C	70	M	67	N	50	54	102	92	62	80	68	58	5	6	5	8	14	16	12	13
8	187982D	65	M	60	Y	78	72	120	124	70	77	63	60	9	6	10	11	16	14	12	13
9	209595D	57	M	57	N	61	65	108	105	72	85	60	86	8	2	2	3	11	13	11	11
10	305723C	65	M	82	Y	59	57	59	72	53	65	57	59	7	3	3	4	15	16	14	16
11	165368A	52	M	55	N	81	77	93	93	55	73	62	66	4	8	8	10	14	14	14	14
12	414531C	62	M	57	N	57	67	98	97	64	78	74	69	3	2	4	3	11	12	12	14
13	657564A	54	M	58	Y	57	72	82	87	68	78	66	59	6	2	6	4	11	11	9	11
14	175489D	59	M	74	Y	61	65	80	89	62	79	70	75	5	0	0	1	14	15	13	13
15	228043D	42	M	76	Y	57	85	94	95	65	64	71	62	7	4	6	6	17	16	14	16
16	226886D	57	M	57	Y	75	78	94	98	79	85	73	78	9	7	5	6	14	13	17	18
17	178432D	53	M	79	N	85	95	108	114	70	72	60	63	3	0	6	7	16	15	15	20
18	191577D	55	M	54	Y	106	118	119	123	68	82	62	60	7	3	3	3	10	12	9	11
19	209666D	72	M	61	Y	72	75	104	105	87	78	97	72	4	1	5	5	12	12	12	14
20	222839D	66	M	73	N	78	85	100	97	66	82	56	58	11	5	5	1	12	11	11	11
21	233852D	30	M	59	Y	75	78	91	84	73	63	74	75	3	1	3	4	13	14	12	13
22	430915C	58	M	53	Y	86	91	95	96	71	59	59	77	7	1	2	4	11	11	11	12
23	227457d	67	M	83	N	62	67	113	109	60	70	65	68	7	2	5	5	16	11	13	13
24	229080D	63	M	55	N	52	54	100	104	67	83	58	66	4	5	6	6	12	13	12	13
25	239742D	57	M	70	N	89	84	98	90	90	80	67	70	4	2	3	5	13	12	13	13
26	233149D	53	M	60	N	84	95	99	96	83	93	75	59	4	3	5	6	11	12	10	11
27	819102C	59	M	70	Y	78	77	100	96	54	65	66	72	4	6	4	5	16	16	12	15
28	011465D	50	M	76	Y	104	91	94	93	111	86	63	57	8	4	5	6	15	15	15	16
29	240240D	70	M	62	Y	53	52	69	83	66	73	70	66	8	8	9	3	14	12	14	17
30	008063D	47	F	50	N	86	104	95	87	86	60	71	57	2	3	2	1	13	12	12	14
31	976823B	53	F	68	Y	59	75	86	82	57	64	67	49	8	6	9	8	17	17	17	18
32	205160D	60	F	43	N	64	68	92	104	60	58	55	60	5	3	4	2	14	14	13	15
33	175924D	59	M	65	Y	54	53	85	97	74	70	76	76	6	4	5	4	15	10	11	12

S.NO	ETCO2(MM/HG)				PaCO2(MM/HG)				EtCO2-PaCO2 GDT				FiO2(%)			
	PRE		POST		PRE		POST		PRE		POST		PRE		POST	
1	29	26	27	27	34.6	30.9	32.5	32.5	5.6	4.9	7.5	7.5	1	0.7	0.7	0.7
2	32	26	35	33	37.1	31	41.1	38.3	5.1	5	6.1	5.3	0.7	0.7	0.7	0.7
3	31	31	37	37	35.2	35.9	42.7	43.5	4.2	4.9	5.7	6.5	0.4	0.4	0.7	0.7
4	35	37	37	43	42.7	44	47.5	52.4	7.7	7	10.5	9.4	1	1	0.5	0.5
5	42	32	35	35	45.8	33.6	39.5	40.8	3.8	1.6	4.5	5.8	0.7	0.7	0.7	0.7
6	29	32	36	34	34.4	35	49.4	46.7	5.4	3	13	12.7	0.7	0.7	0.7	0.7
7	27	29	35	39	32.8	34.8	40.9	46.5	5.8	5.8	5.9	7.5	0.5	0.5	1	1
8	27	26	35	34	33	32	40	39	5	6	5	5	1	0.7	1	0.7
9	28	28	34	33	30.4	32.9	40.5	37.3	2.4	4.9	6.5	7.3	0.5	0.5	0.5	0.5
10	23	23	32	32	27.1	27.2	40.2	39.7	4.1	4.2	8.2	7.2	0.45	0.45	1	0.45
11	30	34	30	31	35.4	37.9	39.9	40.6	5.4	3.9	9.9	9.6	1	0.7	1	0.7
12	28	28	33	34	32.8	32.5	43.2	44.2	4.8	4.5	10.2	10.2	0.7	0.7	0.8	0.8
13	36	36	44	42	39.1	40.1	48.4	46.3	3.1	4.1	4.4	4.3	1	0.7	1	0.7
14	35	31	33	38	39.9	35.5	39.5	44.6	4.9	4.6	6.5	6.6	1	0.5	1	0.5
15	28	29	39	37	32.9	31.4	44.1	42.3	4.9	2.4	5.1	5.3	1	0.5	1	0.5
16	32	33	35	34	39.3	40	49	48	7.3	7	14	14	0.8	0.6	0.5	0.5
17	33	35	41	33	32.6	33	48	39.2	1.6	2	7	6.2	0.45	0.45	0.6	0.6
18	39	41	40	41	43.6	42.2	50.2	51	4.6	2.2	10.2	10	1	1	0.7	0.7
19	36	38	31	29	36.3	44.2	51.2	47.7	0.3	6.2	19.2	18.7	0.7	0.7	1	0.5
20	25	27	33	28	29.1	31.3	38.5	33.2	4.1	4.3	5.5	5.2	0.7	0.7	1	0.7
21	32	31	36	34	35.8	34.4	40.6	38.5	3.8	3.4	4.6	4.5	1	0.6	1	0.7
22	36	38	43	38	40.5	41.2	54.8	46.2	4.5	3.2	11.8	8.2	1	0.7	1	1
23	28	29	38	37	34	35	46.5	46.6	6	6	8.5	9.6	0.75	0.75	0.7	0.7
24	28	29	28	29	30	33.1	33.1	34.8	2	4.1	5.1	5.8	0.5	0.5	0.5	0.5
25	30	31	32	34	35.8	36.5	40.8	48.4	5.8	5.5	8.8	14.4	0.7	0.7	1	0.7
26	34	38	32	32	39.2	42.5	40	38.4	5.2	4.5	8	6.4	1	1	1	1
27	33	32	35	34	37.7	36.1	41.5	42	4.7	4.1	6.5	8	0.6	0.6	1	0.6
28	29	29	32	33	34.1	34	39.5	40.5	5.1	5	7.5	7.5	0.6	0.6	0.6	0.6
29	27	28	29	29	31.8	30	36.1	38	4.8	2	7.1	7.8	1	0.6	1	0.6
30	28	28	28	27	28.8	32.6	33.6	32.2	0.8	4.6	5.6	5.2	1	0.7	1	0.7
31	34	35	33	33	41.8	42.9	46.8	50.7	7.9	7.9	13.8	17.7	0.6	0.6	0.6	0.6
32	32	32	30	31	37.1	37.1	37.1	38.7	5.1	5.1	7.1	7.7	0.5	0.5	1	1
33	30	34	34	36	35.8	39.9	41.5	43.2	5.8	5.9	7.5	7.2	0.5	0.5	1	0.5

S.NO	PaO2(MM/HG)				P/F RATIO				TEMP(°C)				PEEP(CmH2O)				DRN(MIN)
	PRE		POST		PRE		POST		PRE		POST		PRE		POST		
1	430.2	316.6	337.5	267.4	430.2	452.2	482.1	382	36.5	37	37	37.1	0	2	2	2	160
2	307.1	339	286.3	225.6	438	484.2	409	322.2	36.5	34.3	37.6	37	1	2	1	2	120
3	199.7	111.9	167.3	161.3	499.2	279.7	239	230.4	36.6	36.3	36.5	36	2	2	4	4	130
4	511.3	505.4	140.6	146.8	511.3	505.4	281.2	293.6	36.3	35.9	36.4	36.2	2	2	2	2	100
5	308	330.9	339	299.3	215.6	472.7	484.2	427.5	35.9	35.5	36.7	36	1	2	2	2	120
6	155.7	291.8	208.5	143.9	222.4	416.8	297.8	205.5	36.5	36	37.1	36.7	2	2	2	2	100
7	198.8	190.3	444.8	369.4	397.6	380.6	444.8	369.4	35.7	36	36.8	36.6	2	2	2	2	70
8	387.3	290	356	270.3	387.3	414.2	356	386.1	35.6	34.8	36.5	36.1	2	2	2	2	115
9	283	225.6	156.5	309	566	450	313	618	35.8	35.5	37	37.9	2	2	2	2	105
10	210	169.8	318.8	140.4	466.6	377.3	318.8	312	35.9	35.6	36	35.6	2	2	2	2	90
11	391	341.3	486.1	309.1	391	487.5	486.1	441.5	36.5	36.7	37.6	36.3	2	1	2	2	160
12	270.4	296.9	398.9	364.2	386.2	424.1	498.6	455.2	35.6	35.7	36.2	35.7	1	2	2	2	120
13	487.9	330.3	339.9	261	487.9	471.8	339.9	372.8	35.7	35.8	36.8	36.3	2	1	2	2	150
14	435.7	200	480.6	250	435.7	400	480.6	500	35.8	35.2	36.1	36.9	1	2	2	2	100
15	380.7	140.5	379	84.2	380.7	281	379	168	36.5	36	37.2	37.5	1	2	2	2	140
16	332	320	308	290	415	533.3	616	580	36.4	36.2	36.3	36.1	2	2	1	2	150
17	126.2	130.1	160.8	148.4	280.4	289.1	268	247.3	36.6	36.1	36	36	1	0	1	1	95
18	573	470	277	250	573	470	395.7	357.1	36	36.3	36.2	36.2	0	0	0	0	160
19	248	251.5	424.9	129.8	354.2	359.2	424.9	259.6	36	35.3	35.9	35.5	0	0	1	1	150
20	353.6	301.8	475.9	296.5	505	431.1	475.9	423.5	35.6	35.4	36	36.1	2	2	1	2	120
21	439.1	146.6	422.8	288.7	439.1	244.3	422.8	412.4	35.8	35.7	36.4	36.2	0	0	0	0	100
22	458.4	340.4	512	494	458.4	486.2	512	494	36.6	35.9	36	36	2	2	2	2	120
23	249	230	157	129	332	306	224.2	184.2	35.9	35.8	36	36	0	0	0	0	120
24	181.7	261.9	268.8	24.3	363.4	523.8	537.6	428.6	34	33.9	35	35.6	1	2	1	1	100
25	223	234	313	260.3	318.5	334.2	313	371.8	34.6	35	35.2	36.2	0	0	0	0	120
26	302.9	349.7	391.8	369.8	302.9	349.7	391.8	369.8	35.4	35	35.9	35.7	0	0	1	0	100
27	251.4	267.9	432.7	185.7	419	446.5	432.7	309.5	36.4	36.2	36.6	36.4	1	2	2	2	80
28	251.4	251.8	145.1	160	419	419.6	241.8	266.6	35.9	35.5	35.8	35.3	1	2	2	2	70
29	464.2	268.7	406	296.6	464.2	447.8	406	494	36.4	36.2	36.6	35.9	1	1	2	2	80
30	256	182.2	342.4	195.4	256	260.2	342.4	279.1	36.7	36.4	36.2	35.7	1	2	2	2	105
31	244.6	229.3	215.3	149.5	407.6	382.1	358.3	249.1	35.9	35.7	36	35.7	1	1	2	2	90
32	199.5	194.3	407.2	341.6	399	388.6	407.2	341.6	35.9	35.7	35.6	35.2	0	1	2	2	90
33	186.9	223.9	412	142	373.8	447.8	412	284	36	36.1	36.5	36.8	1	2	2	2	100

